Clinical review: gastrointestinal bleeding after percutaneous coronary intervention: a deadly combination

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Summary

Background: Managing gastrointestinal bleeding in a patient who has undergone recent percutaneous coronary intervention requires balancing the risk of stent thrombosis against further catastrophic bleeding. Stent thrombosis and severe gastrointestinal bleeding are life-threatening complications.

Aims: To evaluate the risks of gastrointestinal bleeding in patients undergoing percutaneous coronary intervention in relation to anti-platelet therapy and to discuss management of gastrointestinal bleeding in these patients.


Findings: Gastrointestinal bleeding is relatively common after percutaneous coronary intervention. In one study it complicated 2.3% of primary angioplasty, and these patients had a mortality of 10%. Recent registry data of patients experiencing a gastrointestinal bleed reported a mortality of 5.4%. Cessation of anti-platelet therapy carries a high risk of acute stent thrombosis, which has a high mortality.

Conclusions: Individualized specialist gastrointestinal and cardiological management of these patients in a high dependency environment is recommended. Supportive care and proton pump inhibition in combination with judicious use of anti-platelet therapy is likely to provide the best balance of risk.

Introduction

Percutaneous coronary intervention (PCI) has revolutionized the management of coronary artery disease (CAD) and as the indications for this treatment have expanded, problems have arisen in patients with gastrointestinal (GI) bleeding whilst receiving dual anti-platelet therapy.

The optimum management of a patient with gastrointestinal bleeding who has recently undergone coronary stent implantation is unclear as there is currently little evidence base available. GI bleeding carries a high morbidity and mortality, especially in patients with ischaemic heart disease and left ventricular dysfunction. Yet for these same patients anti-platelet therapy is regarded as essential in preventing stent thrombosis, a potentially life-threatening condition carrying a mortality of up to 80%. In this article, we review the risk of patients developing a significant GI bleed and the available...
evidence to determine a management strategy of dealing with these patients in the context of recent percutaneous coronary intervention (PCI) and the need for ongoing anti-platelet therapy.

The number of patients treated with percutaneous coronary intervention is increasing year on year, tackling increasingly complex problems and patients with multiple co-morbidities. There were 73,692 PCI procedures carried out in the UK in 2006, with an in-hospital mortality of 0.74%.1

Percutaneous coronary intervention now challenges coronary artery bypass grafting as the revascularization procedure of choice for the management of stable angina in patients with multi-vessel disease and is the default strategy in most patients with single or two vessel coronary artery disease. The treatment for acute coronary syndromes (ACS) has also been revolutionized over the past decade achieving significant morbidity and mortality reduction by early angiography and revascularization by PCI.5 Furthermore, primary percutaneous coronary intervention (PPCI) is now becoming the established reperfusion therapy for acute ST elevation myocardial infarction (AMI), superseding thrombolysis due to improved short and medium term clinical outcomes. A total of 3930 patients with AMI in England and Wales patients were treated by PPCI in 2006, and this is likely to increase. In the Czech Republic, 93% of patients with AMI are treated with percutaneous intervention, and 1% receives thrombolysis. The majority of acute physicians and general practitioners will come into contact with patients treated with PCI.

Risk of bleeding during or after PCI

Bleeding has now emerged as one of the most common complications of percutaneous coronary intervention in contemporary practice.5 It occurs at a reported rate in 3.2–9.1% of patients recruited to randomized trials of adjunctive pharmacology during PCI.3–6 The site of bleeding is most frequently related to the femoral access site (52–73% of events). However a significant minority of bleeds, unrelated to arterial access, occur including GI bleeds, intra-cerebral bleeds and large haemoglobin drops without a clinically obvious bleeding site.7

At the time of percutaneous intervention in patients with a recent acute coronary syndrome, there is an associated 3.9% risk of any cause major bleeding (defined as life-threatening bleeding requiring transfusion of ≥2 U of packed red blood cells, or resulting in an absolute decrease in haematocrit of ≥10%, or death, or haemorrhagic/subdural haematoma). Data from 24,045 patients found that the incidence of bleeding complications was highest in patients with ST elevation myocardial infarction (STEMI), with an incidence of 4.8%, 4.7% in non-STEMI and 2.3% in patients with unstable angina.7 A recent study of PPCI reported an incidence of GI bleeding of 2.3%, with a mortality of 10%.8

Patients at high risk may be identified from their baseline demographics and clinical presentation.7 Predictors of bleeding risk are increasing age, female sex, previous bleeding and renal impairment (P<0.01). The GRACE study found that bleeding from any site is an independent predictor of in-hospital death with an odds ratio (OR) of 3.5.7

When any significant bleeding occurs, regardless of source, early discontinuation of anti-thrombotic and anti-platelet agents is usually required, increasing the risks of further ischaemia, infarction, repeat procedures and stent thrombosis. Indeed, rates of Q and non-Q wave MI in published studies are 3- to 5-fold higher in patients with bleeding complications compared to controls. In ACUITY-PCI, stent thrombosis rates were 3.4% in patients with bleeding and 0.6% in patients without bleeding.8

The role of anti-platelet and antithrombotic therapy in patients undergoing percutaneous coronary intervention

A haemorrhagic milieu is present peri-percutaneous coronary intervention as a consequence of the drive to decrease ischaemic complications, endangering susceptible individuals. Peri-procedurally patients are anticoagulated with intravenous heparin or bivalirudin (a direct thrombin inhibitor). Patients at high risk of thrombotic or ischaemic complications are treated with an infusion of a glycoprotein IIb/IIIa (GP) inhibitors, such as abciximab, eptifibatide and tirofiban. In the setting of PPCI these have been shown to reduce absolute mortality at three years follow up by 3.1%.9 GP inhibitors offer protection from major ischaemic events in high risk ACS patients but there is no mortality benefit to their use outside of these patients e.g. in stable CAD. The consequent use of GP antagonists is associated with an increase in GI bleeding.7,10

After the first 24–48 h after percutaneous coronary intervention, two major challenges affect the long-term clinical results: in-stent re-stenosis and stent thrombosis. Coronary artery stents were developed to reduce acute recoil and vessel closure but resulted in less re-stenosis, a natural healing process after the endothelial damage caused by angioplasty. The two main types of stent are bare metal and drug-eluting stents. Drug-eluting stents (DES) were
developed to inhibit the healing response to stent implantation after PCI and has successfully reduced the risk of in-stent restenosis.\textsuperscript{11,12}

The risk of stent thrombosis without anti-platelet therapy is uncertain, but is likely to be around 20\% of the basis of historical data, which has been reduced to 0–2\% with aspirin and clopidogrel dual combination therapy.\textsuperscript{4} Stent thrombosis can be catastrophic and carries a very poor prognosis.\textsuperscript{13} Initial dual anti-platelet regimes were typically for 1 month in the bare metal stent (BMS) era then a continuation of chronic aspirin therapy thereafter. However, due to early concerns of potential delayed endothelization of DES and thus potential thrombogenicity, prolonged dual anti-platelet therapy was extended and recently the European Society of Cardiology guidelines have advocated the use of twelve months of dual treatment compared with 4–6 weeks for BMS.\textsuperscript{14} Short courses of dual anti-platelet therapy are now uncommon due to the increasing use of drug-eluting stents (current usage 50–70\% of PCI cases in the UK), and secondly the recommended 12-month treatment with aspirin and clopidogrel after all acute coronary syndromes regardless of the stent used at the time of PCI.\textsuperscript{15}

Despite dual anti-platelet treatment stent thrombosis still occurs with an incidence of 0–2\% by six months.\textsuperscript{16} Clinical trials comparing BMS and DES have found a similar rate of stent thrombosis,\textsuperscript{16} although outside trials it is higher,\textsuperscript{17} reflecting the increased complexities of cases treated in ‘real world’ settings. Twelve months after PCI there may be a higher rate of stent thrombosis in patients treated with DES compared to BMS.\textsuperscript{18} Recent data suggest an incremental risk of stent thrombosis of 0.6\% per annum after one year with first generation DES compared to BMS.\textsuperscript{18} Prolonged dual anti-platelet therapy beyond one year is even being suggested and prescribed in increasing number of patients.

The incidence of stent thrombosis declines with time but long-term monotherapy with aspirin in patients with the first generation drug-eluting stents is mandatory. This is emphasized by a report of ten patients from one centre who experienced stent thrombosis over a year after stent implantation when aspirin was stopped (average 15.5 days, standard deviation 6.5 days).\textsuperscript{19} There are anecdotal reports of thrombosis occurring over 2 years after stent implantation\textsuperscript{20,21} when all anti-platelet therapy is stopped. Therefore stent thrombosis remains a concern after PCI, is associated with a high mortality rate,\textsuperscript{13} has mandated at least life-long monotherapy and sometimes dual anti-platelet therapy.

### Risk of gastrointestinal bleeding in patients on dual anti-platelet therapy

Anti-platelet agents increase all types of gastrointestinal bleeding and the majority of life-threatening bleeds are upper gastrointestinal. Aspirin is directly ulcerogenic with no consistent dose–response effect.\textsuperscript{22} Clopidogrel is not directly ulcerogenic, and has a lower reported incidence of gastrointestinal bleeding compared with aspirin [all GI bleeds 1.99\% vs. 2.66\% (\textit{P}<0.002), severe 0.49\% vs. 0.71\% (\textit{P}<0.05)].\textsuperscript{23} The current American Heart Association guidelines recommend that clopidogrel be used in those patients who require anti-platelet therapy but who are aspirin intolerant.\textsuperscript{24} In a recent population-based case–control study, combined usage of dual anti-platelet therapy has increased by 425\%. The odds ratio for a serious upper GI haemorrhage (defined either as melaena, or as a subnormal haemoglobin, or need for transfusion together with a potential bleeding source in the stomach or duodenum, excluding varices) were 1.8 (95\% CI 1.5–2.1) with Aspirin therapy alone, 1.9 (95\% CI 0.6–2.1) with clopidogrel alone and 7.4 (95\% CI 3.5–15) on both agents,\textsuperscript{25} illustrating the GI bleeding risk with these agents. However, only 13 patients out of 1443 had gastrointestinal bleeding in the dual therapy group.

Comparison of the trials of anti-platelet regimens is difficult as the severity of bleeding was determined by the treating clinician rather than defined and end points vary. GI bleeding encompasses both upper and lower sources, and the latter are less likely to be affected by anti-platelet treatment. The risk of bleeding is lower with clopidogrel as compared with aspirin,\textsuperscript{23,26} and the risk of bleeding on aspirin combined with clopidogrel is higher than aspirin alone.\textsuperscript{15,27} (Table 1)

General risk factors for gastrointestinal bleeding have been determined and may help patient selection, especially in elective cases for percutaneous coronary intervention. Major factors include increasing age, previous ulceration, helicobacter pylori infection, use of non-steroidal anti-inflammatory drugs and co-morbidity including sepsis and mechanical ventilation.

Intriguingly statins appear to lower the risk of gastrointestinal bleeding after acute coronary syndromes.\textsuperscript{30} Statins have been shown to increase myocardial prostaglandin production, and it is possible that this occurs in the gastric mucosa. Whether statins have a preventative effect on GI bleeding has not been studied but prescription is generally ubiquitous in patients with CAD for secondary prevention.
**What is the mortality risk of gastrointestinal bleeding?**

Upper gastrointestinal bleeding outside the setting of percutaneous coronary intervention is associated with significant mortality. In one study in the UK of 4185 cases the mortality was 14%, but with advances in medication and endoscopic techniques a more recent study found the mortality was 5.4%. The risk of recurrent bleeding and death can be predicted from the patients condition on admission combined with endoscopic findings—the Rockall Score (Table 2). A score of less than 3 carries an excellent prognosis, whilst a score of greater than 8 predicts a high risk of death. This is a well-validated scoring system, although a later study suggests that with advances in treatment that the risk of re-bleeding is over-predicted.

After percutaneous coronary intervention, a patient over sixty years old with coronary artery disease treated with PCI at any time point in the past scores at least 3 on the Rockall scoring system if he has a GI bleed, and therefore carries an increased mortality risk.

Ulcer prophylaxis in the critically ill has become commonplace, but is not routine for patients taking dual anti-platelet therapy. Agents used include H₂ receptor antagonists, proton pump inhibitors (PPI) and sucralfate. Misoprostol is the only agent convincingly shown to reduce the risk of non-steroidal anti-inflammatory-induced GI Bleeding, but commonly causes diarrhoea. There is little comparative evidence to differentiate between these agents. We found no data on the use of prophylaxis following PCI, and this would be worthy of a prospective randomized study. Patients with healed ulceration who are treated with aspirin and esomeprazole have a lower incidence of further gastrointestinal bleeding compared with clopidogrel monotherapy. It should be noted that all patients in this study were either not colonized with helicobacter pylori, or it had been successfully eradicated prior to participation in this study.

**Table 1** A comparison of the incidence of Gastrointestinal bleeding in clinical studies with anti-platelet regimes

<table>
<thead>
<tr>
<th>Study</th>
<th>%</th>
<th>%</th>
<th>Number of patients</th>
<th>Odds ratio (95% CI)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meta-analysis: Aspirin (50–162.5 mg/day) vs. placebo</td>
<td>2.3</td>
<td>1.5</td>
<td>49917</td>
<td>1.59 (1.4–1.81)</td>
<td><em>P&lt;0.0001</em></td>
</tr>
<tr>
<td>Meta-analysis: Aspirin (162.5–1500 mg/day) vs. placebo</td>
<td>3</td>
<td>1.4</td>
<td>16060</td>
<td>1.96 (1.51–2.43)</td>
<td><em>P&lt;0.0001</em></td>
</tr>
<tr>
<td>'CAPRIE' Aspirin 325 mg vs. Clopidogrel—Severe GI haemorrhage</td>
<td>0.71</td>
<td>0.49</td>
<td>19185</td>
<td>1.45 (1.00–2.09)</td>
<td><em>P&lt;0.05</em></td>
</tr>
<tr>
<td>'MATCH' Aspirin (75 mg) and Clopidogrel 75 mg vs. Clopidogrel 75 mg—all life-threatening bleeds including gastrointestinal</td>
<td>1.4</td>
<td>0.6</td>
<td>7578</td>
<td>2.46 (1.48–4.10)</td>
<td><em>P=0.0005</em></td>
</tr>
<tr>
<td>'MATCH' Aspirin (75 mg) vs. Clopidogrel—all major bleeds including gastrointestinal</td>
<td>1.12</td>
<td>0.29</td>
<td>7578</td>
<td>3.87 (1.99–7.53)</td>
<td><em>P&lt;0.0001</em></td>
</tr>
<tr>
<td>'CURE' Aspirin (75–325 mg) + Clopidogrel vs. Aspirin (75–325 mg) life-threatening gastrointestinal bleeds</td>
<td>1.3</td>
<td>0.7</td>
<td>12562</td>
<td>1.79 (1.25–2.56)</td>
<td><em>P=0.0018</em>*</td>
</tr>
<tr>
<td>'Asp 80 mg and Esomeprazole vs. clopidogrel 75 mg alone—recurrent ulcer'</td>
<td>0.6</td>
<td>8.1</td>
<td>320</td>
<td>0.07 (0.01–0.56)</td>
<td><em>P=0.001</em></td>
</tr>
<tr>
<td>'Asp 80 mg and Esomeprazole vs. clopidogrel 75 mg alone—lower GI bleed'</td>
<td>4.4</td>
<td>4.3</td>
<td>320</td>
<td>0.99 (0.34–2.88)</td>
<td><em>P=0.98</em></td>
</tr>
</tbody>
</table>

*This study compared aspirin with a proton pump inhibitor against clopidogrel without a proton pump inhibitor. *Calculated using Fishers exact test. **Calculated using Chi-squared test. Odds ratios calculated (if not available in original paper) by using data from original paper and calculated using Peto odds ratio.

**Treatment of GI bleeding**

**Proton pump inhibitors**

Intravenous infusions of high-dose Omeprazole has become standard therapy for ulcer-related bleeding and have been shown to reduce re-bleeding rates following a randomized controlled trial of 240 patients with bleeding or visible vessels comparing placebo with intravenous omeprazole post endotherapy. Re-bleed rates were reduced from 22.5%
transfusion requirements and need for surgery were also reduced in patients with visible vessels or adherent clot. Once the ulcer is healed and H. pylori eradicated, treatment with aspirin and a PPI has a lower risk of GI bleeding than clopidogrel without a PPI. A recent randomized study of patients with gastrointestinal bleeding treated with endoscopic haemostasis and a PPI found no significant difference in recurrence of GI bleeding between those patients who continued aspirin compared with those who placebo (18% vs. 12%), although there were more vascular events in those taking placebo.37 All cause mortality at eight weeks was also greater in patients who ceased aspirin (14.1% vs 1.6%, p = 0.008).

Endoscopy and endotherapy

Early endoscopy and multi-modality endotherapy are increasingly the norm in the management of upper GI haemorrhage. Registry data of patients with GI bleeding shows that early endoscopy is associated with lower re-bleeding and mortality (OR 0.31, 95% CI 0.11–0.91).32 Although patients with MI are perceived to be at higher risk of complications, this is not the case for haemodynamically stable patients. A case–control study of 200 patients who underwent gastroscopy within 30 days of a MI found the complication rate was 7.5% compared with 1.5% in the controls.38 The complications were largely confined to haemodynamically unstable patients and most were cardio-respiratory, in particular hypotensive episodes (5.5%).

There is limited comparative evidence to choose between endotherapeutic techniques. Where there is active bleeding or a visible vessel, common practice would be dual therapy with a combination of large volume (10–20 ml) epinephrine injection (1 : 10 000), and either thermal therapy with a heater probe or mechanical treatment with clips. Whilst sub-mucosal injection of epinephrine into ulcers can be detected in the circulation,39 there are no case reports of associated arrhythmias.

Endotherapy should be combined with proton pump inhibitor to reduce the risk of re-bleeding and mortality. A randomized trial of 156 patients consisting of intravenous (IV) Omeprazole with or without endotherapy found that endotherapy with IV Omeprazole reduced the absolute risk of re-bleeding by 11% (95% CI 1.7–19.8%, P = 0.01) compared with PPI alone.40 A Canadian observational registry of 1869 patients with GI bleeding, found patients with GI bleeds who were treated with a PPI (85% of patients) had less re-bleeding regardless of endoscopic findings, resulting in a significant mortality reduction (Odds ratio 0.18, 95% CI 0.04–0.8).32 Overall, 6.5% of patients required surgery. The authors concluded that endotherapy and PPI use were independently associated with a decreased mortality in patients whose gastroscopy showed stigmata suggesting

Table 2: Rockall scoring system for risk of re-bleeding and death after admission to hospital for acute gastrointestinal bleeding33

<table>
<thead>
<tr>
<th>Variable</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>0</td>
</tr>
<tr>
<td>Shock</td>
<td>1</td>
</tr>
<tr>
<td>Co-morbidity</td>
<td>2</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>3</td>
</tr>
<tr>
<td>Major SRH</td>
<td>4</td>
</tr>
</tbody>
</table>

Each variable is scored and the total score calculated by summing the scores.

SRH: stigmata of recent haemorrhage.
there was a high risk of re-bleeding. Angiography and embolization of re-bleeding lesions are alternative treatments, but published series are relatively small.

**Investigation of the lower gastrointestinal tract**

Colonoscopy may be undertaken at slightly higher risk in patients with recent myocardial infarction. In a case–control study of 100 patients who underwent the procedure within thirty days of MI with no serious complications related to the procedure. However, given the high (1/769) perforation rate reported in UK practice the attendant risk of laparotomy should be considered. Barium enema is probably lower risk, and CT (computed tomography) or MR colonography is now widely available, so imaging as a first line approach is a rational choice. Anti-platelet agents may not substantially increase the risk of bleeding during therapeutic colonoscopy.

**Cessation of anti-platelet agents after PCI**

Data from retrospective observational studies found a high mortality in patients who stopped anti-platelets to undergo (non-cardiac) surgery immediately after percutaneous coronary intervention. The most likely cause of death was stent thrombosis, although post-mortem or angiographic evidence was not always available. The risk of death within three weeks of PCI was high, between 3.5% and 32%. and a high proportion had stopped anti-platelets (75–86%). In one study, it was reported that only one out of 20 patients who continued ticlodipine or clopidogrel at the time of surgery (5%) died, emphasizing the importance of anti-platelet therapy. Seven weeks after PCI there were no deaths in patients undergoing surgery. The importance of continuing clopidogrel is illustrated by registry data which found discontinuation was the greatest risk factor for stent thrombosis (hazard ratio 90; 95% CI 30–270; P<0.001). A prospective observational study of patients who underwent PPCI found that at eleven months follow up, 7.5% of patients who discontinued clopidogrel within 30 days had died compared with 0.7% who continued therapy (P<0.0001, adjusted hazard ratio 9.0, 95% confidence interval 1.3–60.6).

These data suggest that wherever possible, anti-platelet therapy must be continued. Continued anti-platelet therapy and concurrent anti-ulcer treatment would seem logical in the context of recent stent implantation and upper GI bleeding. There is only one trial (n=9) where patients were treated with cimetidine and continued aspirin despite the presence of active peptic ulceration and fourteen out of fifteen ulcers healed. Proton pump inhibitors have not been studied in this group of patients but may offer similar or better responses.

Within the first 30 days after percutaneous coronary intervention, the risk of stent thrombosis in the presence of dual anti-platelet therapy is at its highest (maximum 1.8%), and without anti-platelet agents the incidence and mortality may be as high as 86%. This risk must be balanced against the risk of further or continued GI bleeding, with the unquantifiable risk of continued anti-platelet drugs.

**Suggested management of significant GI bleeding after PCI**

Our suggested management is based on current practice and supportive data rather than randomized trial evidence (Figure 1). We define significant bleeding as requiring red cell transfusion or causing haemodynamic instability. Close liaison between cardiologist and gastroenterologist is essential. In the light of the United Kingdom National Confidential Enquiry into Patient Outcome and Death (NCEPOD) report into patients undergoing gastroscopy these patients should be monitored in a high-dependency environment, with invasive haemodynamic monitoring to aid fluid management in unstable patients, and critical care advice should be sought.

Hypovolaemia should be corrected as it risks hypotension resulting in organ failure and stent thrombosis. In line with guidance after a myocardial infarct, patients should be transfused to keep the haemoglobin greater than 9 g/dl, and anticoagulation should be reversed. The Rockall score which includes shock, increasing age, co-morbidities and endoscopic factors should be used to stratify an individual’s risk.

Heparin, Bivalirudin or Abciximab infusions should be stopped if these infusions are still active. Continuation of oral clopidogrel is recommended, as it less likely to cause acute erosions than aspirin. Unless ongoing haemorrhage occurs, anti-platelet therapy should not be reversed with platelet transfusions which provide fresh active platelets, capable of clumping and are likely to increase risk of stent thrombosis. Intravenous high-dose proton pump inhibition should be commenced.

Early gastroscopy should be carried out by an experienced team once the patient is resuscitated.
Supplemental oxygen should be given and monitoring of pulse oximetry, blood pressure and electrocardiography is mandatory. NCEPOD recommends anaesthetic advice should be considered. Sedation should be used with caution because of the risks of hypotension and respiratory compromise.

Further use of anticoagulant and anti-platelet therapy should be decided on an individual basis in the light of endoscopic findings. The risk of mortality from discontinued anti-platelet therapy may be felt to be higher than the risk of gastrointestinal bleeding (low Rockall score ≤ 4). However, in patients at high risk of re-bleeding, e.g. visible vessels, the risk of anti-platelet treatment cannot be quantified easily but if the patient has a high Rockall score (≥ 5) we would recommend cessation of aspirin initially (due to its ulcerogenic properties), and clopidogrel for forty-eight hours whilst administering an intravenous PPI. Some patients will require surgical management of their GI bleeding and we would recommend that anti-platelet therapy be continued peri-operatively unless there is concern regarding peri-operative recurrent bleeding and further treatment.

Patients experiencing severe lower gastrointestinal bleeding may be evaluated acutely with CT angiography or digital subtraction angiography. The utility of colonoscopy in active lower GI bleeding has not been firmly established.

### Summary

Gastrointestinal bleeding after stent implantation presents a serious threat to patients due to the competing risks of gastrointestinal haemorrhage and stent thrombosis. Currently there are no guidelines and little evidence of how best to manage this situation. It is clear that these patients are at high risk of morbidity/mortality both from the bleeding itself and the consequences of achieving optimum haemostasis by cessation of anti-platelet therapy and the resultant risk of stent thrombosis resulting in MI and death. Close combined management between gastroenterologist and cardiologist is advocated to optimize patient outcomes. Registry studies of these areas may be helpful in the future to determine and hone the best management strategy.

### References


41. Cappell MS. Safety and efficacy of colonoscopy after myocardial infarction: an analysis of 100 study patients and 100 control patients at two tertiary cardiac referral hospitals. Gastrointest Endosc 2004; 60:901–9.


